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DEVICES FOR PROTECTION AGAINST NEGATIVE ACCELERATION

Part 1. Centrifuge Studies

H. O. SIEKER, CAPTAIN, USAF (MC)

JUNE 1952

WRIGHT AIR DEVELOPMENT CENTER

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DEVICES FOR PROTECTION AGAINST NEGATIVE ACCELERATION

Part 1. Centrifuge Studies

H. O. Sieker, Captain, USAF (MC)

June 1952

Aero Medical Laboratory
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Wright Air Development Center
Air Research and Development Command
United States Air Force
Wright-Patterson Air Force Base, Ohio

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FOREWORD

This report presents the final phase of a collaborative research project studying the problem of protection against the physiological effects of negative acceleration. It was undertaken by the following group: H. O. Sieker, Capt., USAF (MC), O. H. Gauer, M. D., J. P. Henry, M. D., and E. E. Martin, Capt., USAF. The project is identified by Research and Development Order No. R-695-69 entitled 'The Physiology of Negative Acceleration' and was assigned to the Acceleration Section, Biophysics Branch, Aero Medical Laboratory, Research Division. The group acknowledges the strong support and encouragement given by Colonel A. P. Gagge, Colonel R. H. Blount, Colonel H. M. Sweeney and Dr. H. E. Savely. The vigorous technical assistance of Mr. R. U. Whitney, R. F. Managan, T/Sgt., and C. S. Bender, A/1c, was of the greatest value. Finally, the group wishes to express its appreciation to the airmen and officers who were subjects for the various studies.

ABSTRACT

Previous experimental work has shown that negative acceleration is tolerable within certain physiological limits. This report summarizes studies in which human subjects were exposed to negative acceleration with and without protection. The tolerance limit for negative acceleration in unprotected subjects in the upright seated position was found to be 2.5 g. The acceleration was limited to this level by the subject's discomfort and the bradycardia noted in the electrocardiogram. At 3 g, in addition to marked discomfort, conjunctival hemorrhages and cardiac asystoles were noted. When the subject in the upright seated position was protected by means of counterpressure about the head and neck, the tolerance to negative acceleration was increased to 5 g. The tolerance limit for negative acceleration was found to be 4 g in the negative-g aspect of the USAF prone position bed.

PUBLICATION APPROVAL

Manuscript copy of this report has been reviewed and found satisfactory for publication.

Robert H Blount

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SECTION I

INTRODUCTION

A previous study (1) on animal and human subjects has shown that negative acceleration is tolerable with certain physiological limits. Negative acceleration had been avoided by pilots because of the fear of cerebral hemorrhage, mental confusion, and unconsciousness. Experiments done by Armstrong and Heim (2) on animals confirmed the fear of cerebral hemorrhage. However, in 1937, Franklin (3) suggested that the head-down position was not as serious as previously thought, because the brain was protected by being encased in a rigid box. Rushmer, Beckman, and Lee (4) more recently carried out a group of experiments which showed that the veins and probably the minute vessels are almost perfectly protected against sudden changes in intravascular pressure by simultaneous change of the same magnitude in the cerebrospinal fluid pressure. They pointed out, however, that although the arteries received some protection, they were not completely protected. Lombard and his associates (5) exposed goats to 7 negative g without evidence of arterial hemorrhage. One animal in that series "showed a subdural hemorrhage which was slight and of dubious origin." Gamble and Shaw (3) repeatedly exposed dogs to 7 negative g without any evidence of subarachnoid hemorrhage but two animals showed subdural hemorrhages. It seemed possible that the extraneous factors of asphyxia and trauma introduced by the technique of the experiment may have been responsible for these findings.

In more recent studies, Henry and others from the Aero Medical Laboratory (1) found no evidence of cerebral hemorrhage in a large series of goats and dogs exposed to 10 negative g for 3 to 30 seconds, if severe oxygen lack and trauma were avoided. Increases in cerebral intravascular pressure up to 250 mm Hg have been observed in humans during shock therapy without evidence of cerebral hemorrhage (6). The tolerance in humans to negative acceleration lasting for more than 5 seconds has been reported to be 3 g when the subject is in the upright seated posture (1). At 3 negative g, the pressure in the minute vessels of the conjunctiva and sinus mucosa ranges from 50 to 100 mm Hg and pain becomes excessive. These pressures, however, are less than 1/5 the pressure developed in the cranial vessels of the unprotected goat exposed to 15 negative g without evidence of cerebral hemorrhage.

In a previous report (1), it has been suggested that the mental confusion and unconsciousness which have been reported in negative acceleration may be related to reflex cardiac arrest. Jongbloed and Noyons (8) noted in experiments on rabbits that the pressure in the carotid artery increased when the animal was exposed to centrifugal force in the headward direction. Electrocardiograms of these animals showed extrasystoles and bradycardia during exposure to the acceleration. They found that vagotomy or denervation of the cardiac sinus eliminated the cardiac slowing. Ryan, Kerr, and Franks (9) later confirmed Jongbloed's findings. They demonstrated periods of asystole up to 9 seconds and pointed out that in normal subjects the results could not be distinguished from symptoms of carotid sinus hypersensitivity as reported by Weiss and Baker (10). Gamble and others from the Aero Medical Laboratory (11) have confirmed these studies. They point out that with cardiac arrest there is failure of brain perfusion and unconsciousness occurs after 9 to 10 seconds of asystole. Following a period of unconsciousness there is usually a period of confusion of varying duration. In addition, there is the possibility that a reflex of the central type reported by Weiss and Baker (10) may cause unconsciousness without concomitant vasomotor or cardiac symptoms when the carotid area is stimulated. Although it is true that exposures of one minute or more to high negative accelerations will lead to cardiovascular collapse with unconsciousness or death in animals, such prolonged accelerations are not contemplated in man.

Methods of increasing the tolerance to negative acceleration have been discussed in a previous report (1). In these preliminary experiments mechanical pressure in the form of Ace bandages wrapped about the heads and necks of animals was an effective means of preventing the local

edema and pulse slowing that occurred when the animals were submitted to headward centrifugal force without this means of protection. Shaw and Henry (12), using the neck-sealing pressure helmet from the partial-pressure suit (13, 14), found that the venous pressure in the supra-orbital vein could be reduced to near zero levels if pressure was applied to the head and neck with the glottis closed during exposure to negative acceleration. The pressure helmet was used by these workers as a means of counterpressure in human subjects exposed to negative accelerations as great as 5 g for 10 seconds. The symptoms due to vascular engorgement of the head and neck were prevented or minimized by this means. Moreover, the counterpressure about the carotid sinus area prevented the bradycardia found in unprotected subjects exposed to headward centrifugal force.

A second means of protection which was suggested in earlier work was to reduce the heart-to-head distance by changing the position of the pilot. Studies of the venous pressure in the head region showed that in the semi-prone position the hydrostatic column length is decreased and the pressure as measured in the supra-orbital vein should be less than 1/2 that in the upright seated position at a given acceleration. From this it was postulated that the semi-prone position should give protection up to 5 negative g (1).

The purpose of this paper is to report further studies. The average tolerance to headward centrifugal force in the unprotected upright seated position was determined in a series of human subjects. The acceleration was limited by the subjective complaints of the individual and the objective findings noted during and at the end of the exposure to negative g. For this same group of laboratory and flight personnel, the pressure helmet from the partial-pressure suit was then used as means of protection. The tolerance with this form of protection was determined in the same manner as in the unprotected state. The body position of the United States Air Force experimental prone position bed was also used as a means of increasing the tolerance, and was compared with the upright seated position with and without the use of a pressure helmet during negative acceleration.

SECTION II

STUDIES OF NEGATIVE ACCELERATION IN UNPROTECTED AND PROTECTED HUMAN SUBJECTS

Negative Acceleration in Unprotected Subjects

Forty-four individuals, both laboratory and flight personnel, were exposed to negative acceleration in the upright seated posture on the human centrifuge. The subject was placed on his side with his head outward so as to assume a negative-g position during acceleration. He was held in a modified conventional type seat with shoulder harness and seat belt. Each subject was instructed to relax and breathe normally during each exposure to centrifugation. Subjective and objective findings were noted at the end of each run. A modified lead II of the human electrocardiogram was taken with the conventional electrodes; 'left arm' taped over the upper left chest, 'right arm' over the upper right chest, and 'left leg' over the lower left thoracic region. This was done in order to follow the rate and regularity of the pulse.

All of the subjects were exposed to 1 and 2 negative g for 10 seconds. Twenty-three of the individuals were familiarized with negative acceleration unprotected and then exposed to the test acceleration with protection. In this group, the acceleration was limited to 2 negative g to serve as a control for the same subject exposed to higher accelerations with protection. Twenty-one subjects were exposed to headward centrifugal force to find the average tolerance in the unprotected upright seated position. Nineteen of these subjects were able to tolerate 2.5 g for 10 seconds. Five of the 19 subjects were exposed to 3 g for 10 seconds. The acceleration was limited by marked discomfort associated with vascular engorgement of the head and neck and by a marked bradycardia noted in the electrocardiogram. The frequency of the various complaints and objective findings are summarized in Chart I.

CHART I

Acceleration	1g	2g	3g
	% of subjects		
Uncomfortable fullness and pressure in head and neck	40	90	100
Bradycardia	30	90	100
Headache	0	0	50
Asystole	0	0	50
Conjunctival hemorrhage	0	0	40
Difficulty in breathing during acceleration	0	0	40
Diminished vision	0	0	40
Sinus pain	0	0	30
Ear pain	0	0	20

Frequency of symptoms and findings in subjects in the unprotected upright seated position during exposure to negative acceleration.

The most frequent subjective finding was fullness and flushing of the face and neck with a shift cephalad of the soft tissues of the face (cheeks, lower eyelids and lower lip). Every subject had some degree of discomfort with these symptoms. With an increase in the acceleration, in each case there was an increase in the discomfort. The unpleasantness of flushing and fullness of the face and the head and the shift of soft tissues was one of the factors which limited the tolerance to negative acceleration in almost all the subjects to 2-1/2 g for 10 seconds. With termination of the acceleration, this discomfort subsided. Redness and fullness of the face and neck as well as distortion of the face due to shift of soft tissues was observed in all the subjects during centrifugation.

Although all of the individuals had discomfort from the foregoing symptoms, there were particular areas which were uncomfortable or painful in some subjects. One-half of the people exposed to 3 g noted a generalized headache for one-half to two days following the exposure. About 20 percent of the subjects noted pain in the frontal and temporal areas of the head. With an increase in the acceleration, the pain increased in severity and became a limiting factor for the tolerance in these persons. One-fourth of the group noted pressure and pain in the region of the maxillary and frontal sinuses at the higher negative accelerations. Some also noted increased mucous discharge from the nose. These symptoms subsided gradually after the acceleration was stopped. It should be pointed out that in some of these subjects an upper respiratory infection was present which would increase the discomfort associated with the vascular engorgement of the sinus areas. Three of the subjects complained particularly of pressure or pain in the ears at 2-1/2 g. This symptom subsided at the termination of the acceleration, and physical examination at this time showed only slight injection of the ear drum.

One-fourth of the series noted blurring of vision at 2-1/2 to 3 g. This was usually associated with tearing of the eyes and a tendency for the lower lid to cover the eye. No instance of red-out was noted. All of the subjects showed some degree of injection of the conjunctiva which subsided shortly after the run was terminated. Two individuals, however, sustained conjunctival hemorrhages following exposure to 3 g for 10 seconds. For this reason also, the exposure of the unprotected human was limited to 2-1/2 g when the discomfort and bradycardia became fairly marked. Fundiscopic examination following the unprotected runs showed no change in the retina or retinal vessels.

No instance of unconsciousness was noted in this series. One person, however, reported mental confusion during an exposure to 3 g during which he also had cardiac asystole for 9 seconds. All of the subjects showed a slowing of the pulse during negative acceleration. With greater acceleration for a given individual, there was usually a greater slowing of the pulse to 40% of the control pulse rate at 2 to 2-1/2 g. In addition to the subject who had cardiac asystole for 9 seconds during an exposure to 3 g, two subjects exposed to 3 g for 10 seconds showed asystole for 2 to 3 seconds. This was another reason for limiting the exposure in the unprotected upright seated position to 2-1/2 g. Cardiac extrasystoles also were noted in several subjects.

Four of the subjects noted moderate difficulty in breathing at 2-1/2 and 3 g. This was primarily a difficulty in filling the lungs during inspiration because of the headward shift of the diaphragm and abdominal contents. It was not severe enough or of long enough duration to cause any respiratory embarrassment.

Previous studies have pointed out that the danger of cerebral hemorrhage is extremely small in human subjects exposed to negative accelerations up to 5 g. Moreover, cerebral dysfunction, change in lung function, and cardiac damage are not limiting factors at low levels of negative acceleration. In this series of unprotected subjects in the upright seated posture, the tolerance was 2.5 g. This was limited by the subject's discomfort and the bradycardia which developed. Individuals exposed to 3 g had marked discomfort, episodes of cardiac asystole and mental confusion, and conjunctival hemorrhages. In order to increase the tolerance, methods of protection were devised. Their effectiveness will be discussed in the remainder of this report.

The Use of Counterpressure as a Means of Protection During Negative Acceleration

Twenty-nine individuals, both laboratory and flight personnel, were familiarized with the use of the pressure helmet and the negative-g valve (USAF M-7) (15) for protection in negative acceleration, (Figure 1.) One size of the pressure helmet from the partial-pressure high altitude suit was used on all subjects, but an attempt was made to vary the size of the neck seal to fit the individual. The neck seal was reinforced by an elastic bandage or by the collar of the partial-pressure suit. The helmet was held in place by a harness attached about the groins or by the attachments on the partial-pressure suit which was worn in some of the experiments.

Based on previous venous pressure recordings from the supra-orbital vein at various negative accelerations, a pressure of 25 mm. Hg. per g was used in the helmet during exposure to headward centrifugal force (1). The pressure in the first fifteen subjects was delivered through a manually operated solenoid with an increase of pressure of 25 mm. Hg. per g. However, this did not allow a gradual increase in pressure as the g increased, and a negative-g valve (USAF M-7, Figure 2) was developed (15) and used by the last fourteen people in the series. This valve permitted the subject to breathe oxygen normally at safety pressure while at one positive g. With the onset of negative acceleration, the valve delivered a graded pressure of 25 mm. Hg. per g with a gradual fall in pressure when deceleration occurred.

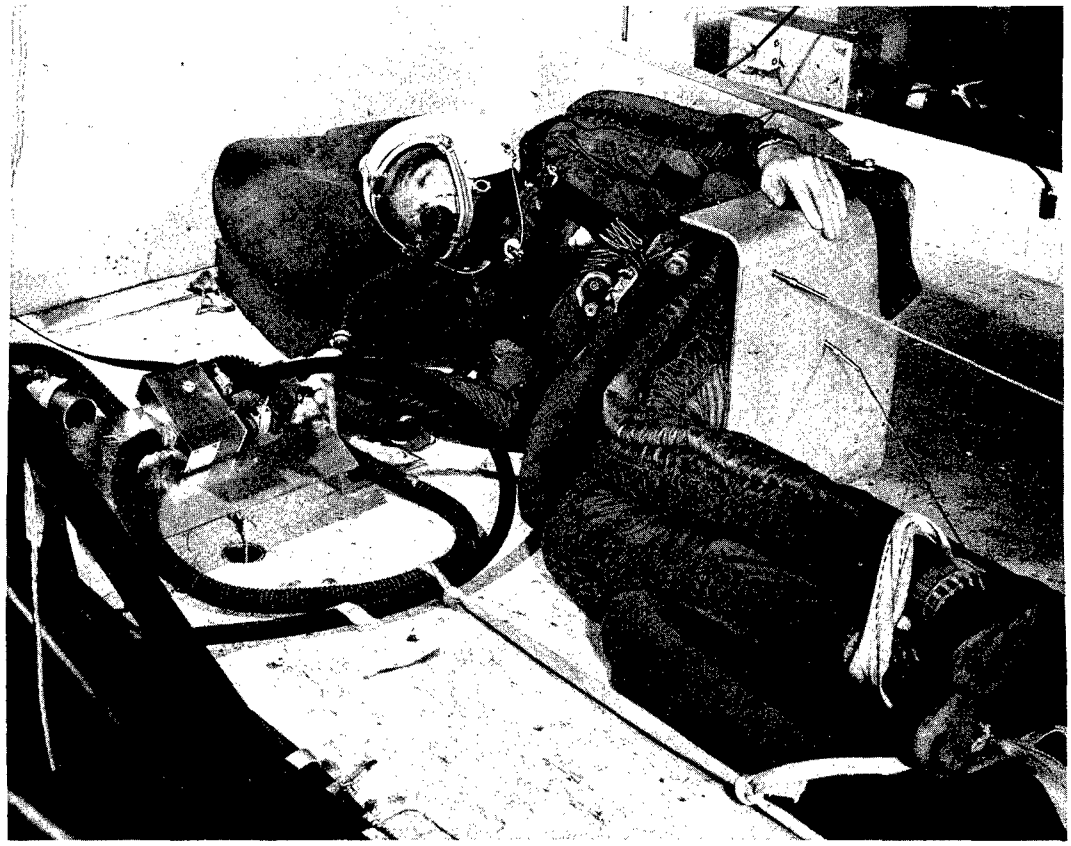

Fig. 1 The neck-sealing pressure helmet and the partial-pressure high altitude suit on a subject in the modified conventional seat mounted on the centrifuge platform. The subject is positioned so that he will be exposed to negative acceleration. The USAF M-7 negative-g valve is mounted on a swivel plate so that it will be activated by negative acceleration.

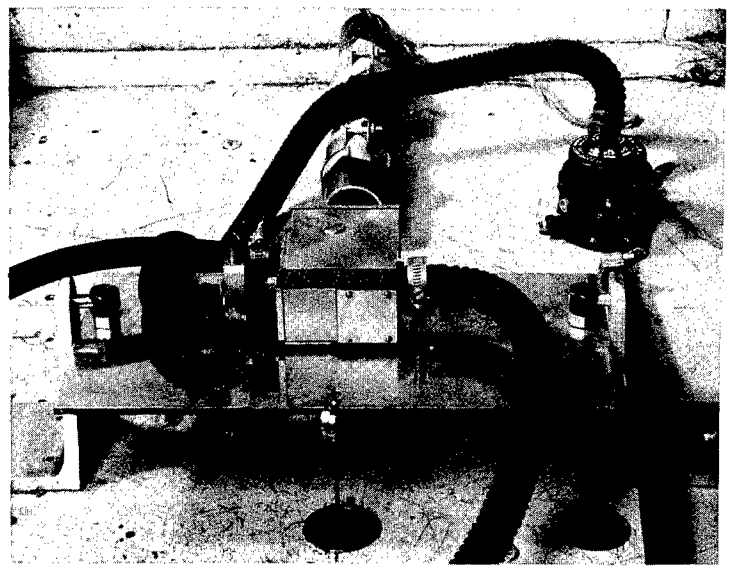

Fig. 2 A close-up view of the USAF M-7 negative g valve shown in Figure 1.

The upright seated position was used in the negative-g aspect on the centrifuge in the same fashion described in Section II-A. The subject was instructed to relax during the run and to note any unpleasant sensations as well as to note his visual, auditory, and mental states. During the period of exposure to acceleration, the subject held his mouth open so that the pressure was equalized throughout the helmet, pharynx, Eustachian tube and middle ear. Each individual was also instructed that he would be most comfortable if he held that volume of air in his lungs which left his chest in the relaxed position. Helmet pressure was sensed by a Statham pressure transducer which actuated a Heiland galvanometer in a recording oscillograph. A modified lead II of the electrocardiogram was used to follow the rate and regularity of the pulse. The acceleration was also recorded so that a record similar to Figure 3 was obtained in each instance. All of the exposures to negative acceleration were limited to 10 seconds, and all individuals were exposed to 1 and 2 g without protection as a control before using the protective equipment.

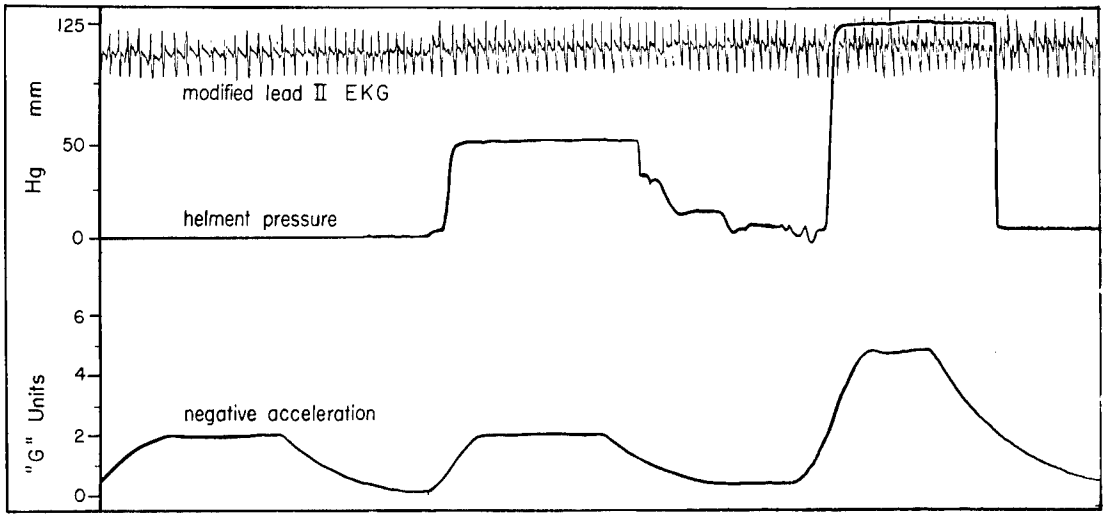

Fig. 3 An actual record of exposures to negative acceleration with and without the pressure helmet as a means of protection. Note that pulse rate is slower at 2 g without protection than at 5 g with protection.

All of the subjects tolerated 3 negative g with less discomfort and with less pulse slowing than 2 negative g without protection. Fourteen of this group were trained in the use of the equipment up to only 3 g because they were to test the equipment in an aircraft which is limited to this acceleration. In the group of fifteen subjects exposed to headward centrifugal force with protection, eleven persons found 4 g less or about as uncomfortable as 2 g without protection. Four found 5 g with protection no more uncomfortable than 2-1/2 g without protection.

In Chart II are presented the complaints and findings which limited the tolerance with the pressure helmet. The sensations of fullness and pressure in the head and neck and shifting of the soft tissues of the face were the most frequent complaints, but at a given acceleration they were much less severe in the protected state than in the unprotected state. Most subjects received 2 to 2-1/2 g protection against these discomforts when using the pressure helmet. As was true in the unprotected state, there were particular areas of the head and neck which were more uncomfortable than other areas. A few subjects noted vague generalized headaches for as long as one-half day following the exposure to 3 to 5 g for 10 seconds. Sinus pain and pressure were absent or less marked up to 5 g when protected than at 2 to 2-1/2 g without protection. Only one person noted sinus pain at 4 g. Eight subjects, however, experienced ear pressure and pain at negative accelerations from 2 to 5 g. The pain usually increased in severity as the acceleration increased and several subjects were stopped at 4 g. In most of these cases, upper respiratory infections

CHART II

Acceleration	1g	2g	3g	4g	5g
	% of subjects				
Uncomfortable fullness and pressure in the head and neck	0	10	40	50	80
Bradycardia	60	50	50	40	30
Ear pain	0	10	30	50	50
Diminished vision	0	0	10	20	30
Headache	0	0	0	0	30
Difficulty in breathing during acceleration	0	0	0	0	30
Sinus pain	0	0	0	0	0
Asystole	0	0	0	0	0
Conjunctival hemorrhage	0	0	0	0	0

Frequency of symptoms and findings in the upright seated position when the subjects were exposed to negative acceleration and protected by the full pressure helmet.

were not present. In an attempt to alleviate this discomfort, several subjects were exposed to the same acceleration with 5 to 10 mm. Hg. less pressure per g. In this situation, the ear pain was less but the protection was limited by increased discomfort from vascular engorgement of the head and neck and by a greater bradycardia. Individuals who were able to hold their breath with their mouth open and glottis closed did not notice this pain.

Three of the subjects noted some blurring of vision at 3 to 5 g with protection. This was primarily related to tearing of the eyes and to a tendency for the lower lid to cover the eye. It was of no greater magnitude than that found at 2-1/2 g without protection. Conjunctival injection was much less marked at each acceleration with counterpressure than without counterpressure. No instance of conjunctival hemorrhage was noted in accelerations up to 5 g when the subjects were protected. Fundiscopic examination was entirely negative following the runs.

The electrocardiogram was followed in order to compare pulse slowing in the protected and unprotected states. These studies showed that in each case, the bradycardia at any acceleration was less with the pressure helmet than without the helmet. Figure 3 shows an actual record with pulse rate, helmet pressure, and acceleration. It can be seen that the pulse rate is slower at 2 g without protection than at 5 g with protection. In Figure 4, the average percent fall of pulse rate is compared in the unprotected and protected state at each g. The average percent slowing of the pulse is greater at 1 and 2 g without counterpressure than at accelerations up to 5 g with counterpressure. Moreover, it should be pointed out that at higher negative accelerations (3 to 5 g) with protection, there is actually an increase in pulse rate. No instance of cardiac asystole was noted even at 5 g, when the subject wore the pressure helmet. The discomfort from vascular engorgement of the head and neck was the factor that limited the acceleration with counterpressure to 5 g in most of the subjects.

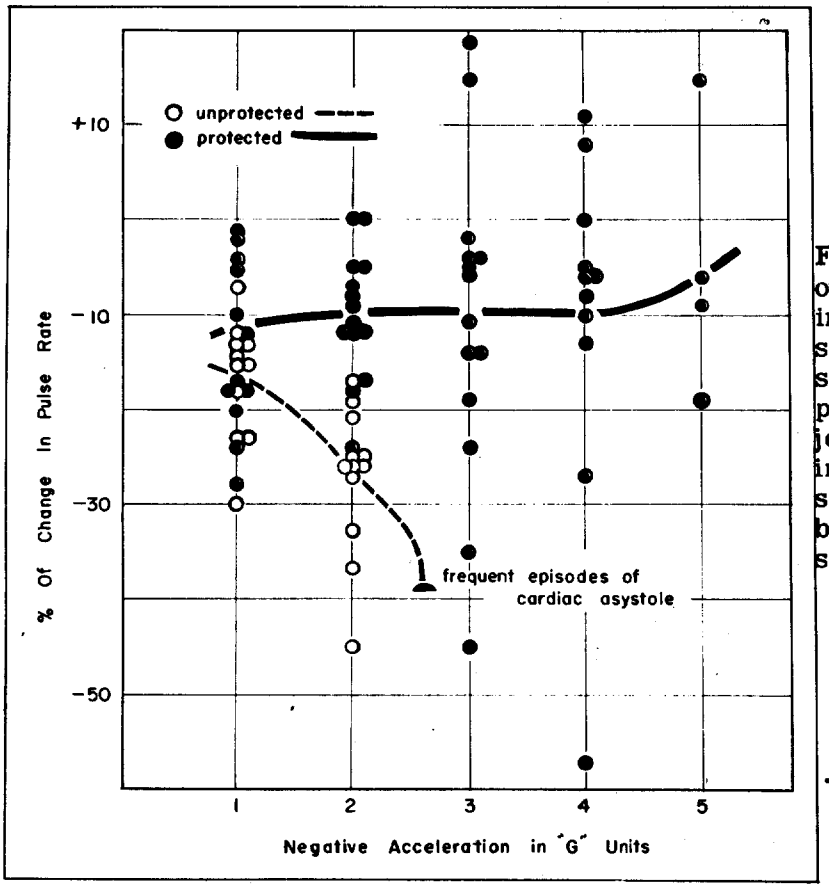

Fig. 4 The average percent slowing of the pulse rate at each acceleration in the unprotected subjects is represented by the broken line while the solid line represents the average pulse slowing in the protected subjects. It should be noted that with increasing acceleration, the protected subjects did not show an increasing bradycardia, while the unprotected subjects did.

None of the subjects had difficulty in holding his breath while pressure was applied in the helmet. However, at 3 to 5 g, all of the group were more conscious of a headward shift of the diaphragm and abdominal contents. Some of the individuals held their breath at the end of full inspiration and, with headward displacement of the diaphragm and abdominal contents, pressure and pain were noted in the chest. When the subject held his breath with the chest at a relaxed volume, this discomfort was avoided or minimized. At 3 to 5 g when the pressure helmet was used, there was also an increase in the discomfort from pressure on the shoulders as the weight of the body was forced against the harnesses. Two of the subjects who were exposed to 4 and 5 g were found to have petechiae in the skin in the region around the shoulder harness and over the arms, shoulders, and chest. No petechiae were noted under the helmet.

By the use of counterpressure about the head and neck, individuals have been protected to 5 g. The tolerance is limited to this acceleration by the fact that the subject's discomfort is comparable to the discomfort at 2 to 2-1/2 g unprotected. Even at 5 g, when the subject was protected, the average pulse slowing was only 10% of the resting pulse. In the unprotected state at 2-1/2 g, the slowing was 30 to 40% of the resting pulse with instances of cardiac asystole at 3 g.

The USAF Experimental Prone Position as a Means of Protection Against Negative Acceleration

The USAF experimental prone position bed of the type pictured in Figure 5 (16) was used as a means of protection against negative acceleration. In this position which is not true prone but semi-prone, the head is at an angle so that the line of vision is 20^{o} below the horizontal, and the trunk is at about a 20 degree angle with the horizontal. The thighs are at a 40 degree angle, and the lower legs are flexed at a 35 degree angle with the horizontal. When this prone position bed is used in positive acceleration, there is free movement of the hands, arms, feet, and head which is supported by a counterbalanced head support. For the negative acceleration experiments, the form of the posterior surface of the body while in the prone position bed (pictured in Figure 5) was made. This form was mounted on the platform of the human centrifuge to support the subject while he was exposed to negative acceleration. The subjects were further held in position by a harness over the shoulders and about the waist, (Figure 6.)

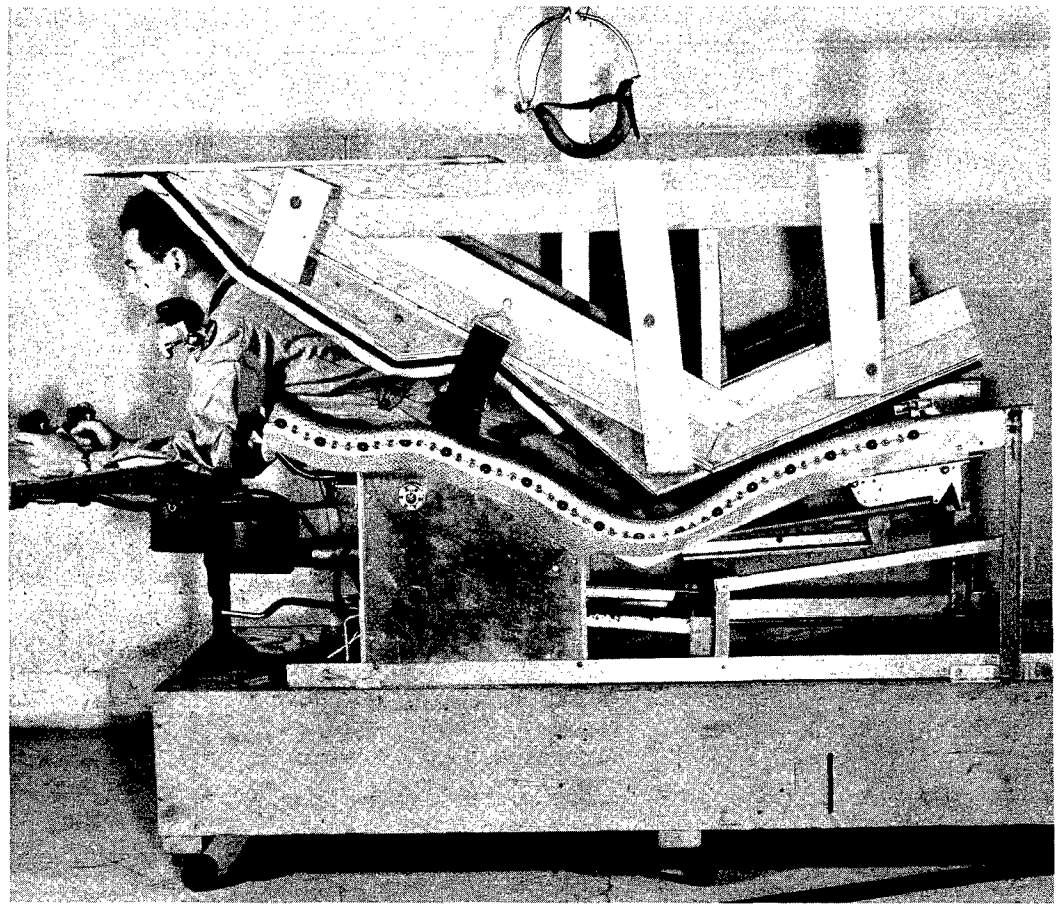

Fig. 5 Subject in the USAF experimental prone position bed with the form for negative acceleration fitted to the posterior surface of his body

Venous pressure was measured in the head region in two subjects while they were exposed to negative acceleration. The pressure was measured in the supra-orbital vein by an indwelling venous needle which was connected to a Statham pressure transducer. The transducer activated

a Heiland galvanometer in a recording oscillograph. In addition, 14 individuals were exposed to headward centrifugal force in the same manner in order to find the tolerance to negative acceleration in a larger series of human subjects. Subjective and objective findings including those made with the modified lead II of the electrocardiogram were noted. Ten-second exposures to acceleration were carried out at 1 g increments to 4 g.

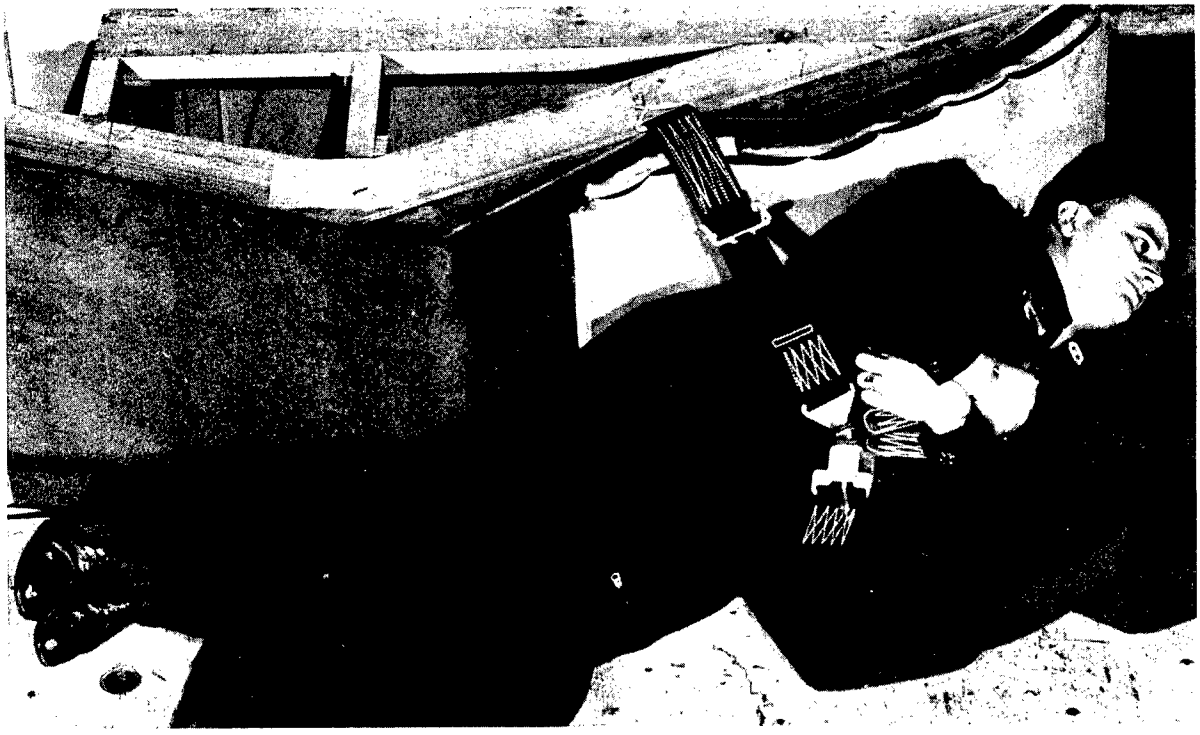

Fig. 6 Subject positioned on the centrifuge platform against the form for the study of the negative acceleration aspect of the semi-prone position bed.

Venous pressure measurements were made to find the acceleration at which the increase in venous pressure in the head region was comparable to the increase in venous pressure in the same area when the subject was seated upright at 2 to 3 g. The results are shown in Figure 7. It can be seen that the venous pressure in the supra-orbital vein rises in an almost linear fashion. The venous pressure in the upright seated position at 2 to 2-1/2 g was found to be 80 to 90 mm Hg (1). In the semi-prone position at 4 g, the venous pressure in the same vein was found to be 75 to 85 mm Hg. On the basis of these experiments, it was postulated that the semi-prone position would give about 2 to 2-1/2 g protection. A larger series was then tested in order to find the tolerance using only subjective and objective findings other than supra-orbital venous pressure as the limiting factors.

All of the fourteen subjects exposed to headward centrifugal force in the semi-prone position were able to tolerate 4 g. The tolerance was limited by complaints of congestion and fullness in the head and neck and pressure and pain in the chest and abdomen from headward and dorsal shift of the diaphragm and thoracic and abdominal viscera. The electrocardiographic changes of bradycardia, asystole and extrasystoles at 4 g were also limiting factors. Chart III summarizes the frequency of these findings at each acceleration.

All of the subjects had fullness and pressure in the head and neck regions during the exposure to negative acceleration. This increased in severity as the acceleration increased, and was

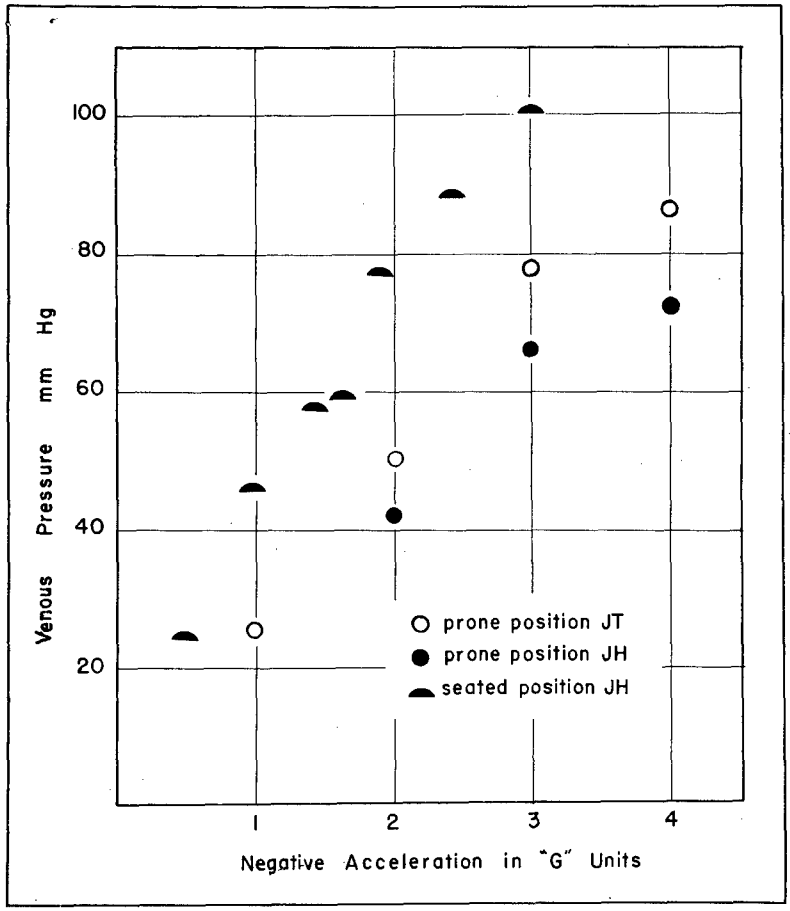

Fig. 7 The venous pressure in the supra-orbital vein as compared in the unprotected upright seated position and in the semi-prone position during negative acceleration.

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CHART III

Acceleration	lg	2g	3g	4g
		% of subjects		
Uncomfortable pressure and fullness in the head and neck	50	70	90	90
Bradycardia	30	90	100	100
Difficulty in breathing during acceleration	0	10	30	70
Pain and pressure in chest and abdomen	0	10	30	40
Diminished vision	0	10	10	10
Ear pain	0	10	0	20
Asystole	0	0	0	30
Headache	0	0	0	0
Conjunctival hemorrhage	0	0	0	0

Frequency of symptoms and findings in subjects during negative acceleration in the semi-prone position.

severe enough at 4 g in most of the individuals to stop at this acceleration. About one-half of the subjects also noted discomfort from the shift of the soft tissues of the face including the cheeks, lower eyelids, and lower lip. Pain was not the main complaint, although the pressure and fullness of the head and neck in some individuals bordered on pain. Several subjects also noted a vague dull headache during the exposure to 4 g and for a short period of one-half to two hours after the exposure. Three of the group noted pressure and pain in the sinus areas associated with the generalized pressure and fullness of the head and neck. In every instance, congestion and increased mucous secretion were noted in the nasal passages at 3 and 4 g in this position. It should be pointed out that, as is true in the seated position, an upper respiratory infection will increase the discomfort from exposure to negative acceleration. About 20 percent of the subjects noted pressure of severe degree in the ears at 4 g. In two, this was associated with difficulty in hearing during the exposure to the acceleration.

Ten to twenty percent of the group had blurring of vision at 2 to 4 g. This was associated, as in the sitting position, with tearing of the eyes and a shift of the lower eyelids over the eyes. In every case, there was slight to moderate injection of the conjunctiva after the exposure to negative acceleration. This subsided a short time after the subject left the centrifuge and no instance of conjunctival hemorrhage was noted. Fundiscopic examinations at this time revealed no abnormalities in the retina or retinal vessels.

The electrocardiogram showed a pulse slowing when the subject was exposed to negative acceleration in the prone position. In Figure 8, the percent change in pulse rate in the experimental position is compared at each acceleration with the percent slowing when the subject is in the upright seated posture. It can be seen that at lower negative accelerations the average percent slowing of the pulse is about the same in the prone position and in the upright seated position. At 3 g, the average pulse slowing in the prone position was 42 percent and there were no instances of cardiac asystole. In a smaller series of upright seated subjects, however, there were two instances of cardiac asystole for 3 to 9 seconds. At 4 g, in the prone position, there were 4 instances of cardiac asystole for 3 to 7 seconds. Three of the subjects also showed frequent premature beats at 4 g in this position. Because of the frequent occurrence of asystole and premature beats, the tolerance to negative acceleration in the semi-prone position was limited to 4 g.

Eleven of the 14 subjects noted marked difficulty in breathing at 3 to 4 g in this experimental position. This was primarily a difficulty in inspiration and was associated with a decreased lung volume due to headward shift of the diaphragm and abdominal viscera. Associated with this, most of the subjects complained of pressure and even pain in the chest and in the region of the diaphragm at 4 g. This was also one of the factors limiting acceleration in this position.

From the studies of venous pressure, it was postulated that the semi-prone position would enable the subject to tolerate 4 to 5 negative g. The discomfort from vascular engorgement of the head and neck was no greater at 4 negative g in the semi-prone position than at 2-1/2 g in the upright seated position. However, the chest pressure and respiratory difficulty at 4 g in this position were more marked than in the upright seated position at 2-1/2 g. At 3 g, asystole occurred in several instances in the upright seated position when the average pulse slowing in the semi-prone position was about 40 percent. However, at 4 g, cardiac asystole occurred frequently in the latter posture. For these reasons, it is believed that 3-1/2 to 4 g is the limit of tolerance to negative acceleration when a semi-prone position of the type discussed is used.

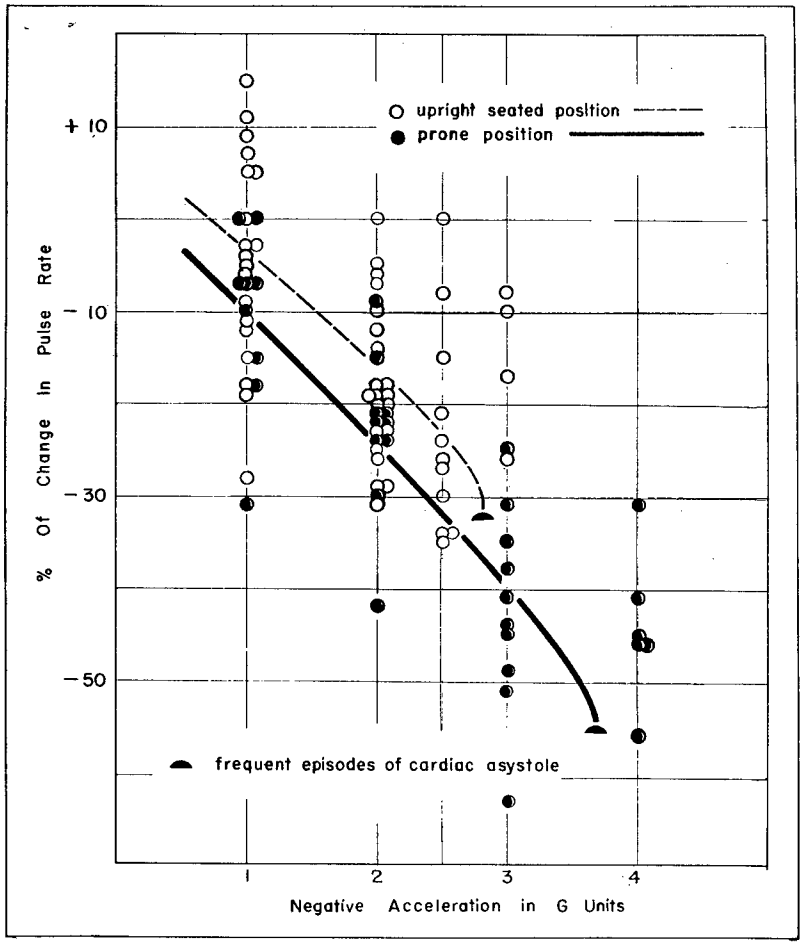

Fig. 8 The average percent slowing of the pulse rate at each acceleration in the unprotected upright seated position is represented by the broken line. The solid line represents the average pulse slowing in subjects in the semi-prone position. It should be noted that periods of cardiac asystole occurred at 3 g in the upright seated position and 4 g in the USAF experimental prone position.

SECTION III

CONCLUSIONS

In a series of unprotected subjects in the upright seated position, the tolerance to negative acceleration was 2-1/2 g. The factors limiting the tolerance to this level were: (1) the discomfort from vascular engorgement of the head and neck and (2) the bradycardia which developed as a result of increased pressure in the vascular system of the cephalic region. The tolerance was further limited to 2-1/2 g by the fact that at 3 g conjunctival hemorrhages, cardiac asystole and episodes of confusion occurred in some of the subjects exposed to this acceleration.

One form of protection tested was the application of counterpressure about the head and neck by means of the pressure helmet from the partial-pressure high altitude suit. With this type of protection, subjects tolerated 5 g for 10 seconds with no more discomfort than 2-1/2 g for 10 seconds without protection. Moreover, at 5 g in the protected subjects there were no instances of conjunctival hemorrhages which were seen in the unprotected individuals at 3 g without protection. It has also been shown that the pulse slowing is no greater at 5 g with protection than at 2 g without protection and there was no instance of the cardiac asystole or mental confusion as was found at 3 g in the unprotected state. It is concluded that suitable protection against negative accelerations up to 5 g can be obtained by use of counterpressure about the head and neck.

A second means of protection used was the semi-prone position, as provided in the USAF experimental prone position bed. The venous pressure rise at head level in this position was the same at 4 g as in the upright seated position at 2 to 2-1/2 g. Although the symptoms associated with vascular engorgement of the head and neck were no greater in this position at 4 g than in the upright seated position at 2-1/2 g, the pressure in the chest and the difficulty in breathing experienced by the subjects was greater. At 4 g in the prone position, cardiac asystole occurred frequently. Because of these findings, the tolerance to negative acceleration in this position was limited to 4 g. At present, protection can best be obtained by means of counter-pressure about the head and neck. This raises the tolerance to at least 5 g or 2-1/2 g above the limit in the unprotected state.

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